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ESTIMATED THYROID IRRADIATION OF UTAH INFANTS DUE TO NUCLEAR TESTS IN NEVADA

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ABSTRACT: Assay of milk for fallout I^{131} from Nevada tests during 1962 indicated an average thyroid dose of about 1.0 rad to the 53,000 Utah infants under 2 years old. Unfortunately, I^{131} had not been measured in milk or people during earlier testing. However, thyroid doses from the earlier tests can be estimated crudely using air beta concentrations and published yields of announced detonations. The number of exposed Utah infants and their estimated average thyroid doses are listed for each year of Nevada testing:

YEAR	INFANTS UNDER 2 YR.	ESTIMATED DOSE (RAD)
1951	40,000	0.4
1952	41,000	3.8
1953*	43,000	6.3
1955	45,000	2.0
1957	47,000	8.6
1958	48,000	1.4
1962	53,000	1.0
*St. George	700	68

Using unverified assumptions, 0-22 thyroid cancers from Nevada I^{131} fallout are predicted for Utah children. Despite uncertainties in these estimates, they indicate that studies for possible delayed effects of radiation should be considered for Utah.

METHODS OF ESTIMATING THYROID EXPOSURE

The problem of estimating thyroid doses from I^{131} when the I^{131} was never measured is very difficult. This difficulty does not decrease the need to obtain the information. Methods for estimating I^{131} dose will be discussed in detail, both to indicate how my estimates were made, and to point out how these estimates could be improved with additional data. Only the I^{131} dose from Nevada testing is considered: the total dose is somewhat larger due to the short-lived iodine isotopes[1] and the I^{131} from Pacific and Russian tests.

I^{131} is a fission product formed in nuclear explosions. It and other products are carried in the air downwind from the point of detonation. When this fallout settles on pastures, the exposed forage becomes contaminated. I^{131} appears in the milk of cattle ingesting I^{131} in their feed. If man drinks this radioactive milk, I^{131} concentrates in and irradiates his thyroid gland. These processes suggest 5 ways for estimating thyroid exposure from I^{131}. They are listed in decreasing order of validity in Table 1 and will be discussed separately.

Table 1
WAYS OF ESTIMATING THYROID EXPOSURE

A. I^{131} in thyroid gland
B. I^{131} in milk
C. Gamma activity in pastures
D. Beta activity in air
E. Fission yield and fallout trajectory

A. I^{131} IN THYROID GLAND

The most direct way to evaluate I^{131} dose to the thyroid is to measure its I^{131} content by γ-ray counting. We measured I^{131} in 24 people during the Utah I^{131} incident of 1962, but unfortunately failed to include infants among this sampling[2,3]. Children 0-2 years old are regarded as most susceptible to I^{131} radiation damage because of (a) the small size of a child's thyroid, (b) its presumed greater sensitivity to irradiation and (c) the long post-irradiation life span during which delayed effects could appear. A drawback to thyroid counting is that specialized equipment is required and only a limited number of people can be evaluated.

B. I^{131} IN MILK

Fresh milk is by far the major source of fallout I^{131} for our population. Milk is more readily sampled and analyzed than are humans. Using the conventional (although perhaps inaccurate) assumptions for the infant of 30% I^{131} uptake in the thyroid, an effective retention half-time of 7.6 days and a thyroid weight of 2 grams; an intake of 58,500 picocuries of I^{131} delivers a dose of 1.0 rad to the thyroid. In the 1962 incident the average intake for consumption of 1 liter of milk per day was 58,000 pc I^{131} from Pendleton's 39 milk farms scattered throughout Utah[2,3]. A somewhat lower intake of 37,000 pc I^{131} (the corresponding infant thyroid dose is 0.63 rad) was obtained by the U. S. Public Health Service for the Salt Lake City milk pool[4,5]*. Unfortunately, I^{131} was not measured in milk or people throughout Utah during Nevada testing prior to 1962. This seriously limits the accuracy of the remaining methods for estimation of I^{131} exposure.

C. GAMMA ACTIVITY IN PASTURES

The arrival of fallout causes an increase in γ-ray "background". I^{131} is but one component of this activity. Fortunately, variations in the I^{131}/total activity ratios can be reduced by correcting the γ-reading to a standard reference time such as 1-day after detonation using the relation[6] that the total activity varies approximately as $(\text{time})^{-1.2}$. The I^{131}/total ratio will still depend on which element was fissioned (U^{235} or Pu^{239}), whether I^{131} selectively holds back or falls out relative to the other fission products (fractionation), and how much of the total

*The Salt Lake City milk pooled sample is made up of equal volumes of milk from each of 14 dairies (of unequal size) which supply milk to Salt Lake City (not the entire state).

activity was due to activation of stable elements by neutron capture (for example, Na^{24} or W^{187}). Using the increased γ-activity on the ground as an index of I^{131} contamination in exposed forage, I^{131} ingested by cattle and subsequently appearing in milk can be estimated. The amount of contaminated feed consumed needs to be known unless the correspondence between γ-activity on the ground and I^{131} activity in the milk can be established for a similar incident under the same farming practices. Gamma measurements were made by others during the 1962 incident. When (if?) these data are released it should be possible to estimate the I^{131} exposure from previous incidents by this method.

D. BETA ACTIVITY IN AIR

The Utah State Department of Health began daily measurements of the gross beta concentration in air at Salt Lake City in 1956. Dr. Grant S. Winn has made these complete records available to me. Usually air was drawn through a filter continuously for about 24 hours. Then the filter pads were beta-counted after a suitable wait (usually 2-6 hours) to allow the radon daughters to decay to insignificance relative to the fission products.

Each peak concentration exceeding 100 picocuries per cubic meter (pc/m^3) was assigned to a preceeding Nevada test. This arbitrary limit of 100 pc/m^3 was low enough to include all important Nevada fallout trajectories at Salt Lake City, but high enough to exclude Pacific and Russian tests. Normal background during periods of no testing was about 1-2 pc/m^3. Published meteorological trajectories for 1957[7] and 1962[5] were particularly helpful in assigning air concentrations to particular tests.

Additional air beta measurements have been made in Utah by other

Table 2

AIR BETA CONCENTRATIONS IN UTAH

All samples were taken in Salt Lake City by the Utah State Department of Health except for the tests Easy (Ogden), Fox (Price), Annie (St. George) and Harry (St. George).

NEVADA EXPLOSION	NAME OF TEST	KILOTON YIELD	HRS BURST TO ASSAY	AIR pc/m^3 AT ASSAY	AIR pc/m^3 AT 24 HRS	HOURS SAMPLED	AIR WTD. AV. pc/m^3
7 MAY 52	EASY*	12	?	20,000*	?		
25 MAY 52	FOX*	11	?	1,000*	?		
17 MAR 53	ANNIE**	16	2.29	147,000	8673	0.59	
			5.13	49,000	7693	5.08	
			9.67	5,000	1680	4.00	
			13.75	4,000	2048	4.17	
			19.97	1,000	802	10.16	2,817
19 MAY 53	HARRY**	32	3.76	4,170,000	450,360	5.33	
			8.04	2,380,000	642,600	3.25	
			11.67	630,000	264,600	4.00	
			15.80	44,000	26,620	4.25	
			21.75	14,000	12,432	7.17	239,564
28 MAY 57	BOLTZMANN	12	129.58	266	2,014		
2 JUN 57	FRANKLIN	0.14	34.08	445	676		
18 JUN 57	WILSON	10	55.37	675	1843		
5 JUL 57	HOOD	74	28.08	498	603		
15 JUL 57	DIABLO	17	32.58	7,090	10,281	2.50	
			33.17	3,000	4,410	2.50	
			35.58	2,440	3,904	2.50	
			40.75	1,887	3,566	5.08	
			52.50	258	660	11.42	3,006
24 JUL 57	KEPLER	10	36.00	644	1,050	7.91	
			55.59	73	200	16.09	480
18 AUG 57	SHASTA	17	35.00	908	1,426	4.84	
			35.75	3,164	5,094	2.83	
			39.83	2,260	4,158	4.00	
			52.42	1,245	3,175	12.33	3,212

*Air concentrations from Ref. 6. Time from burst to assay not given.
**Air concentrations and times are to mid-sampling period; data from Ref 8.

NEVADA EXPLOSION	NAME OF TEST	KILOTON YIELD	HRS BURST TO ASSAY	AIR pc/m^3 AT ASSAY	AIR pc/m^3 AT 24 HRS	HOURS SAMPLED	AIR WTD. AV. pc/m^3
23 AUG 57	DOPPLER	11	54.67	552	1,485		
30 AUG 57	FR. PRIME	4.7	75.08	164	645		
31 AUG 57	SMOKEY	44	74.83	261	1,023		
2 SEP 57	GALILEO	12	75.00	928	3,647		
6 SEP 57	WHEELER	0.2	27.08	114	132		
16 SEP 57	NEWTON	12	26.92	7500	8,625		
23 SEP 57	WHITNEY	19	81.00	137	590		
28 SEP 57	CHARLESTON	12	98.33	212	1,153		
7 OCT 57	MORGAN	8	57.58	878	2,511	6.33	
			78.25	215	888	17.67	1,316
22 OCT 58	SOCORRO	6	73.50	501	1,914		
30 OCT 58	BLANCA	19	100.00	533	2,958		
6 JUL 62	SEDAN	100*	49.00	900	2,115*		
7 JUL 62	L.FELLER II	<20	51.00	101	250		
11 JUL 62	JOHNIE BOY	<20	52.08	16	41		
14 JUL 62	SMALL BOY	<20	50.25	450	1089		
17 JUL 62	L.FELLER I	<20	50.42	11	27		

*Much of the fallout activity from Sedan was due to neutron activated W^{187} (10) which is not a fission product. Less than 30 kilotons of the Sedan yield was from fission(5).

agencies, but thus far I have discovered only two references[6,8] giving some of these results. Further data are needed - especially for the years 1951, 1952, 1953 and 1955.

To allow for different times of arrival, I have adjusted air concentrations to a common reference time of 1 day after detonation assuming a $(\text{time})^{-1.2}$ variation in activity. Unfortunately, reference (6) does not give the time from burst to assay for the Easy (7 May 52) and Fox (25 May 52) shots. Results are listed in Table 2. Times of explosion and kiloton yields are from The Effects of Nuclear Weapons 1962[9].

In some cases a number of air samples were taken serially during a 24 hour period. In such instances each adjusted air concentration was multiplied by the fraction of a day it was sampled. Then these results were added to obtain the weighted average concentration for the entire 24 hour period.

If one assumes a proportionality between beta activity in the air and I^{131} reaching man through the food chain it is possible to crudely estimate thyroid exposures for those years for which air data are available. Results are shown in Table 3.

Table 3

THYROID DOSE ESTIMATES FROM AIR BETA CONCENTRATIONS

YEAR OF TESTS	SUM OF 24 HR. PEAK ADJ. AIR CONC. (pc/m^3)	ESTIMATED AV. INFANT THYROID DOSE (RAD)
1951	?	?
1952	21,000**	5.9
1953*	?	?
1955	?	?
1957	30,450	8.6
1958	4,872	1.4
1962	3,522	1.0***
*St. George	240,000	68

**Time after burst, not given[6], has been assumed by me (perhaps incorrectly) to be 1 day.

***Calculated from measured I^{131} in milk.

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It must be realized that the values in Table 3 are subject to great uncertainties, the greatest of which is probably how well (or poorly) a single air monitor station can sample a fallout trajectory. Contamination of unmonitored parts of the state were undoubtedly both higher and lower than indicated by these estimates. It is unknown how closely the dairy practice in St. George, Utah corresponds to that for the state as a whole. In 1962 every case of high I^{131} in milk could be traced to grazing on contaminated pasture or using contaminated feed. Very little I^{131} appeared in the milk of cattle exclusively eating feed which had been stored prior to the contaminating event [2,3].

Most of the air beta in 1962 was from the Sedan shot [2,3,4,5], but much of the Sedan activity was due to neutron-activated W^{187} [10]. Our measurements of milk collected between the Sedan and Small Boy shots indicated that about 75% of the I^{131} from the July 1962 tests was from Small Boy alone [3]. Normalizing to the 1089 pc/m^3 and 0.75 rad from Small Boy would increase the estimated doses for previous years in Table 3 by a factor of 2.4. On the other hand, if the average intake for the total state is assumed to equal the USPHS value for the Salt Lake City milk pool, all estimated doses in Table 3 should be multiplied by 0.6.

E. FISSION YIELD AND FALLOUT TRAJECTORY

In Table 3 no air beta data were available (to me) for the major milk producing areas of Utah in 1951, 1953 and 1955. Therefore, another method was used to estimate the exposure for these years. I^{131} exposure was assumed proportional to the yields of nuclear devices detonated between 1 April and 31 October of each year. Vegetation eaten by milk cows does not grow in the winter and thus is unlikely to be contaminated with I^{131} from winter shots. The same holds for a number of other fission products and suggests an excellent way to conduct nuclear tests with a minimum of exposure to the population.

In addition to all of the limitations described in the last 2 paragraphs of the preceding section on air beta concentration, one must add uncertainties in the direction of fallout and its rate of descent. Meteorological trajectories (for constant altitude) are available for the tests in 1953[8], 1957[7] and 1962[5], but what we really need are the upper-air fallout (U.F.) trajectories which predict the deposition of fallout along the ground.

Thyroid dose estimates from fission yields are given in Table 4.

Table 4

THYROID DOSE ESTIMATES FROM FISSION YIELD

YEAR OF TESTS	KILOTON YIELD (1 APR - 31 OCT)	EST. AV. INFANT TH. DOSE (RAD)
1951	18	0.4
1952	64	1.6
1953	252	6.3
1955	84	2.0
1957	344	8.6*
1958	57	1.4
1962	?	1.0

These estimates could be recomputed independently if the yields of the five tests of July 1962 were released. The sum of their fission* yields is indicated to be "less than" 110 kilotons[5], but how much less was not given. For what it is worth (which may not be much), Table 4 predicts about 40 kilotons fission yield during July 1962. There may be legitimate security reasons for withholding this information.

* The 8.6 rad dose for 1957 was estimated from the calculated 1.0 rad dose for 1962 and the air beta concentrations for 1962 & 1957 (see Table 3).

* Dunning states that about 1000 kilotons of fission was released prior to 1959 at the Nevada test site[7]. The total (fission + fusion) yield for Nevada tests prior to 1959 was 1036 kilotons[9]. Thus virtually all was fission.

COMPOSITE DOSE ESTIMATES

Combining the results from air beta concentrations (Table 3) and kiloton yields (Table 4) the following dose estimates are presented in Table 5. Populations estimated from 1950 & 1960 U. S. Census and Ref[7].

Table 5

SUMMARY OF DOSE ESTIMATES

YEAR OF TEST	UTAH INFANTS UNDER 2 YR	EST. AV. TH. DOSE (RAD) AIR BETA	YIELD	MEAN
1951	40,000	?	0.4	0.4
1952	41,000	5.9	1.6	3.8
1953*	43,000	?	6.3	6.3
1955	45,000	?	2.0	2.0
1957	47,000	8.6	8.6	8.6
1958	48,000	1.4	1.4	1.4
1962	53,000	1.0	?	1.0
*St. George	700	68	-	68

Agreement between the 1957 dose estimates is a consequence of the way the data was normalized and signifies nothing. The agreement between the 1958 estimates may reflect the fact that the air beta measurements in 1957 and 1958 were made at the same place (Salt Lake City) by the same agency (Utah State Department of Health). However, the 1958 agreement may be fortuitous. A larger difference is seen in the 1952 estimates when the air samples were taken at Ogden and Price by a different agency and the times after detonation were not given[6].

It should be noted that in the 1962 incident individual doses ranged from essentially zero up to at least 14 times the state average[2,3] and similar variations have probably occurred in previous years.

Despite the limited accuracy of these estimates, 5 conclusions are suggested:

(a) The exposure in 1962 was small compared to that during several preceding years.

(b) The worst years seem to be 1953 and 1957.

(c) Many Utah children have received thyroid doses of several rads.

(d) The St. George exposures were sizable.

(e) These dose estimates need to be improved. In particular, field gamma data should be released; air beta concentrations should be made available for 1951, 1952, 1953 and 1955; and if not contrary to the interests of national security, kiloton yields for the July 1962 tests are needed. Perhaps a future "incident" will provide the needed calibration check!

PREDICTED THYROID CANCERS

I realize that any attempt to predict an increase in cancer due to low doses of radiation is subject to great inaccuracy and criticism. This is especially true when the dose estimates are so very approximate. Therefore, it should be understood that the following estimates were made primarily to indicate whether or not an effort to search for increased thyroid cancers would be justified.

Archer and Simpson[11] have tabulated 10 thyroid cancers in 2253 children x-irradiated as infants for "an enlarged thymus" with an average dose of 225 rads, and an average follow-up time of 14.5 years. At present we have no information on how many additional thyroid cancers will develop at later times, ie after 14.5 years. They have calculated that 0.05 "spontaneous" thyroid cancers should have been expected normally in this group during the follow-up period. Assuming a linear relation between dose and incidence*, there should be 1 case per 50,000 rad-children.

* To my satisfaction this relation has neither been proved or disproved adaquately for thyroid cancer.

There is some evidence[12] that thyroid irradiation from I^{131} is only about 1/10 as effective as x-irradiation in producing thyroid cancer in rats, but it is unknown whether this relation also applies to the human infant. The predicted numbers of thyroid cancers in Utah children are tabulated in Table 6 for both a relative biological effectiveness (R.B.E.) of 1 and a R.B.E. of 0.1. These values are compared with the number of "spontaneous" cases expected by age 15 years. Children are arranged by age recognizing that some children were irradiated at age 0-1 and again at age 1-2. Irradiation received at age 2 and older has been assumed arbitrarily (although perhaps incorrectly) to be without effect.

Table 6

PREDICTED THYROID CANCERS IN UTAH CHILDREN

AGE IN 1963	NUMBER OF CHILDREN	EST. AV. TH. DOSE (RADS)	PRED. TH. CANCERS RBE = 1	PRED. TH. CANCERS RBE = 0.1	"SPON."
13	20,000	0.4	0.16	0.02	0.44
12	20,000	4.2	1.68	0.17	0.44
11*	21,000	10.1	4.24	0.42	0.47
10*	21,000	6.3	2.64	0.26	0.47
9	22,000	2.0	0.88	0.09	0.49
8	22,000	2.0	0.88	0.09	0.49
7	23,000	8.6	3.96	0.40	0.51
6	24,000	10.0	4.80	0.48	0.53
5	24,000	1.4	0.67	0.07	0.53
4	25,000	0	0	0	0.55
3	25,000	0	0	0	0.55
2	26,000	1.0	0.52	0.05	0.58
1	27,000	1.0	0.52	0.05	0.60
0	27,000	0	0	0	0.60
*St. George	700	68	0.95	0.10	0.02
Irradiated	250,000	4.4	21.90	2.20	5.57

The predicted total number of fallout-induced thyroid cancers is 22 (for an R.B.E. of 1) or 2 (for an R.B.E. of 0.1). These would be in addition to the 6 "spontaneous" cases expected to develop in this 1/4 million children during their first 15 years. The large number of irradiated children provides a rare opportunity to test the hypothesis that small doses to the infant thyroid from I^{131} are equally as effective rad-for-rad as are large doses of x-rays abruptly given.

On the other hand, if an RBE of 0.1 is closer to the truth, then it will be difficult to separate the radiation-induced cancers from the "spontaneous". One cannot help but wonder what fraction of the so-called "spontaneous" cancers are in fact due to medical x-rays given before or after birth.

If a sufficiently high threshold exists for the induction of thyroid cancer by irradiation, then there may be no cases caused by fallout.

The fact that many children were born when there was no testing within 2 years of birth provides an internal control by which the normal incidence of thyroid cancer in Utah can be established. Also, the uneven distribution of fallout suggests that slightly contaminated areas could serve as controls for those heavily contaminated. Finally, the fact that with other factors being equal, I^{131} dose is proportional to milk consumption suggests that <u>in theory</u> the low milk drinkers could serve as controls for the high milk drinkers. However, from the practical standpoint, the work and uncertainty in estimating individual milk consumptions in retrospect does not appear promising except for limited areas such as St. George which received appreciable doses.

An interesting implication is the prediction that the number of cases at St. George is small compared to that for the state as a whole. It is indeed fortunate that only a few hundred children received high exposures.

Crude as these dose estimates are, they indicate the advisability of studies for possible delayed radiation effects in Utah. To be fruitful such studies should have long-term support and look for other effects in addition to thyroid cancer.

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